137 Body Size and Habitus

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Definition

Body size and habitus describe the physical characteristics of an individual and include such considerations as physique, general bearing, and body build. Historically, attempts have been made to classify humans into discrete somatotypes (mesomorphic—muscular and athletic; endomorphic—rounded and stout; and ectomorphic—tall and thin), and to relate habitus to propensity to disease (e.g., habitus apoplectus). These terms have little clinical relevance today, and body size and habitus can be said to encompass the more quantifiable measurements of height, weight, body proportions, skinfold thickness, and mid–upper arm circumference. These measurements do not have true normal or abnormal values, but must be interpreted in the context of an individual's age, sex, clinical status, and previous measurements. The values can be plotted as a percentile of a reference population or as a percentage of an "ideal" value.

Height is the length from the plantar surface of the foot to the crown of the head. Heights that fall below the 3rd percentile or above the 97th percentile may require investigation.

Weight is the total weight of the body. Weights greater than 120% of "ideal" suggest obesity, while weights less than 70% of "ideal" may indicate severe malnutrition.

Body proportions include the *trunk to limb ratio* and the *arm span.* The trunk, or "upper segment," is represented by the distance from the symphysis pubis to the crown of the head. The "lower segment" is the distance from the symphysis pubis to the plantar surface of the foot and represents the "limb" contribution to total height. At birth the normal upper to lower segment ratio is 1.7 : 1. The legs grow more rapidly than the trunk, and by age 10 the segments are equal and remain so in adults. The arm span is the distance between the tips of the middle fingers with the arms fully extended. In adults the arm span should equal the height.

Mid–upper arm measurements are taken at the midpoint between the acromial process and the olecranon process. *Skinfold thickness* at this site is a measure of subcutaneous fat and is used to estimate total adiposity. Obesity is indicated by a value greater than 23 mm in men and 30 mm in women. Severe depletion of energy stores is indicated by values below the 30th percentile. *Mid–upper arm circumference* is used to calculate *mid–upper arm muscle circumference.* Muscle circumferences less than the 30th percentile suggest severe depletion of protein stores.

Technique

Accurate measurement of height requires a firm horizontal surface, a measuring tape attached to a true vertical surface, and a movable block at right angles to the vertical surface that can be brought down to make contact with the scalp. The unsteady base and flexible bar of most office scales limit their accuracy. The subject should stand in bare feet, with the heels together and the back as straight as possible. The outer canthus of the eye should be in the same horizontal plane as the external auditory meatus when the headpiece is brought down to the crown of the head.

Body weight should be measured on beam scales with nondetachable weights. Spring scales are not sufficiently accurate. The scale should be calibrated to zero. The patient should be dressed in light clothing without shoes. Nonambulatory patients can be weighed in bed or wheelchair scales. Patients' estimates of their weights and heights are not reliable.

Arm span is most easily determined by doubling the distance between the sternal notch and the tip of the middle finger of an extended arm. The lower segment measurement is taken with the subject standing and is the distance from the symphysis pubis to the floor. The upper segment is calculated by subtracting the lower segment from the total height.

Triceps skinfold thickness is generally measured at the midpoint between the acromial process of the scapula and the olecranon process of the ulna of the left arm, using a skinfold caliper. This point is marked on the posterior side with the patient sitting or standing with the arm hanging loosely at the side. Patients unable to sit may be measured supine in bed with the arm folded across the chest. The skin and subcutaneous tissues should be pinched between the thumb and forefinger 1 cm above the mark and gently pulled away from the underlying muscle. While the grasp is maintained, the calipers are placed over the skinfold at the midpoint mark, left in place for 3 seconds and the value is read. The average of three separate readings is recorded in millimeters. Plastic calipers may not be accurate. The subscapular skinfold is similarly measured 1 cm below the right scapula.

Arm circumference, recorded in centimeters, is also measured at the upper arm midpoint. A measuring tape encircles the arm and is pulled snugly without pinching the skin. Arm muscle circumference is calculated with the formula:

$$\text{Arm muscle circumference} = \text{arm circumference} - \pi\ (\text{triceps skinfold})$$

All values are converted to centimeters.

Basic Science

Nutritional Status

While sophisticated methods are available for determining the relative contributions of water, fat, bone, and protoplasm to total body weight, these methods are too cumbersome for routine clinical use or epidemiologic studies. For clinical purposes, weight, height, and skinfold thickness are used to estimate the degree of adiposity in evaluating for obesity and, along with arm circumference, to estimate depletion of energy and protein stores in the malnourished hospitalized patient.

HEIGHT AND WEIGHT

Body weight alone is a poor measure of adiposity because weight is strongly correlated with height. Clinicians have generally relied on height and weight tables to determine the appropriateness of a patient's weight for his or her height. The 1959 Metropolitan Life Desirable Weight Tables in use for the past two decades were based on actuarial data from the 1959 Build and Blood Pressure Study of insured individuals. The term "ideal weight" was coined by Metropolitan Life to encourage people to keep their weight below the average for the insured population. The ideal weight was that associated with the maximum longevity for each height and "frame size." Guidelines for determining frame size were not given and actually represented an arbitrary division of the population into the low (small frame), middle two (medium frame), and highest quartiles (large frame). In fact, there are few studies assessing the contribution of frame size to weight. The "body mass index" (BMI = weight/height^2) has been promoted as a superior index of relative weight.

The revised 1983 Metropolitan Life Height and Weight Tables are based on the 1979 Build Study of 4.2 million persons insured by 25 U.S. and Canadian life insurance companies over 22 years. The 1983 tables include guidelines for determining frame size from elbow breadth. An estimate of frame size can be made by encircling the patient's wrist at the widest point with the examiner's thumb and index finger. If the fingers overlap, the frame size is small; if they just meet, the frame size is medium; and if the fingers do not meet, the frame size is large.

The insurance tables have their limitations as representations of ideal weight. In fact, the 1983 revised tables no longer carry the term "desirable" in their title. The insurance data represent a sample of a selected group of healthy middle-class individuals. The average weight of the sample is less than the average found in more representative cross-sectional studies.

In general, obesity is readily diagnosed by examination of the naked patient. The height and weight tables, despite their limitations, provide useful reference standards, as several studies have shown an increased mortality with weights greater than 15% above or below the actuarial ideal weights. Their use in determining weight goals should be tempered by clinical judgment. Patients with hyperlipidemia, hypertension, or non-insulin-dependent diabetes mellitus may have an increased percentage of body fat and may benefit from weight loss despite weights within the reference range. There may also be a group of "healthy obese" persons who frequently have strong family histories for obesity and who themselves have been obese in the past, but who have managed to reduce their weight and maintain it at a level where fat cell size is normal and where there are no identifiable risk factors (Callaway, 1984). These persons would be unlikely to benefit from further efforts at weight reduction.

Because of the wide variation in body size, comparison of weight to a reference standard may be less useful in assessing the adequacy of caloric intake than determining the percentage of usual body weight and the rate of weight loss. Weights less than 90% of usual are evidence of significant malnutrition.

SKINFOLD THICKNESS AND ARM MUSCLE CIRCUMFERENCE

Almost one-half of the body's fat is distributed as subcutaneous fat. As the thickness of this layer bears some relationship to total body fat, and as the loss of subcutaneous fat occurs proportionately with dieting, measurement of skinfold thickness is a relatively simple and practical method of determining the extent of fatness. The triceps and subscapular skinfolds are commonly used, and age-specific tables derived from cross-sectional studies provide reference values for each measurement and their sum.

While the presence of obesity is usually obvious with excessive weights, moderate degrees of overweight may be due to increased muscularity (e.g., football players). Skinfold thickness may be used to supplement weight and height measurements. Measurement of body fat is also useful to estimate the duration and severity of inadequate dietary intake as fat is lost slowly in malnutrition.

Mid–upper arm muscle circumference is a reliable index of skeletal protein mass. It does not take into account bone and other nonmuscle tissues below the skinfold or the shape of the arm, but is useful as a comparative measurement for which standards have been developed. The measurement is subject to some lack of reproducibility between observers. In addition, applying standards derived from healthy populations will overestimate protein–calorie malnutrition. Hospitalized patients often have chronic illness that may result in disuse or denervation atrophy and subsequent loss of muscle mass despite adequate protein stores.

Laboratory assessment of nutritional status may include determinations of serum albumin, transferrin, creatinine-height index, total lymphocyte count, and skin tests for cell mediated immunity. These tests all suffer from a lack of specificity. At least one study has shown that a history and physical by a well-trained physician was as reliable in determining the presence of protein–calorie malnutrition as a complete anthropometric and laboratory work-up (Baker et al., 1982).

SPECIAL CONSIDERATIONS IN THE ELDERLY

The changes of aging may complicate the nutritional assessment of the elderly. Most standards are derived from younger populations. The mean height of older age groups is less than the height of younger groups. This can be attributed to genetic and environmental influences on the current aged and to the loss of stature that occurs with aging. The loss of height is primarily due to shortening of the spinal column from thinning of the disks and osteoporosis. Accurate measurement may be difficult to obtain because of kyphosis or illness that precludes standing erect. Loss of height may be an important indicator of osteoporosis. Serial heights are an essential measurement in the elderly.

There tends to be a progressive decline in weight after age 50, accompanied by a decrease in lean body mass and an increase in adipose tissue. Most of the increased fat is deposited around the internal organs. There is some evidence that skinfold measurements on the trunk are more reliable predictors of body fat in elderly males, while extremity measures may be more accurate in elderly females.

Stature

Growth proceeds in a nonlinear fashion with the greatest rate immediately after birth. There is a general deceleration throughout childhood, except for a small mid-childhood growth spurt and a prominent adolescent growth spurt, after which the epiphyses close and maximum height is attained. The regulation of growth is a complex, incompletely understood phenomenon with genetic, hormonal, and environmental influences. Although racial differences exist, it is not clear how much of these differences are environmentally determined. Some of the hormonal influences on growth are outlined below:

- *Thyroid hormone* appears to moderate growth by regulating synthesis and secretion of growth hormone, as well as by a permissive action for the effects of growth hormone. Thyroid hormone is necessary to develop normal adult proportions.
- *Growth hormone* increases chondrogenesis, protein synthesis, and cell profileration. Its growth-promoting actions may be mediated through somatomedins.
- *Insulin* acts primarily to preserve the metabolic homeostasis necessary for growth rather than as a direct growth stimulator.
- *Glucocorticoid* oversecretion can inhibit growth, apparently by a direct effect on target tissues, rather than by inhibiting growth hormone actions or release.
- *Androgens* have a complementary action with growth hormone and are potent growth stimulators in prepuberty. However, they also accelerate epiphyseal maturity. Administration of androgens may lead to early epiphyseal closure and loss of growth potential. Hypogonadism may cause a delay in closure and result in long limb length.
- *Estrogens* have an inhibitory effect on linear growth and a stimulatory effect on epiphyseal maturity. Estrogens may inhibit somatomedin secretion.

Table 137.1
Health Consequences of Obesity

Hypertension	Established relationship between overweight and hypertension; weight reduction reduces BP.
Cerebral vascular disease	2 to 3 times risk of cerebral vascular disease in obese hypertensives.
Premature coronary disease	Marked obesity is associated with increase in CAD, lesser degrees variably associated.
Lipids	Ratios of LDL/HDL and cholesterol/HDL are affected adversely.
Diabetes mellitus	Factor most strongly related to adult-onset diabetes mellitus; weight loss is key to control.
Arthritis (DJD, gout)	Definite association of obesity and gout; association with DJD not proven, but weight loss frequently improves symptoms.
Gallbladder disease	2 to 3 times risk of death by gallbladder disease in obesity.
Pulmonary	Marked obesity may be associated with hypoventilation, hypoxia, and sleep apnea.
Surgical risk	Increased risk with anesthesia, poor wound healing, thrombotic complications.
Psychosocial	Social stigma, discrimination, poor self-image.
Miscellaneous	Propensity for skin infections, varicose veins, endometrial carcinoma.

Note: BP = blood pressure. CAD = coronary artery disease. DJD = degenerate joint disease.

Clinical Significance

Obesity and Malnutrition

If obesity is defined as 120% of actuarial ideal weight, then cross-sectional surveys indicate that 25 to 30% of men and women in the United States are overweight. The prevalence increases with age until middle age, after which it declines. Obesity is more prevalent in women than men, particularly in women of lower socioeconomic status.

Most studies have shown an increased mortality in overweight individuals. Recent studies have shown the minimum mortality around the average weight with excess mortality in those 20% below or 20% above the average weight. In the 1983 revision of the Metropolitan Height and Weight Tables, the ideal weights were adjusted upward 5 to 10% to a "slightly plump" weight. Table 137.1 summarizes some of the health consequences of obesity.

Although obesity results from an intake of calories in excess of energy expended, there are undoubtedly genetic, metabolic, endocrine, and behavioral influences. The onset of obesity early in life results in hyperplasia of fat cells, which may lead to a more intractable form of obesity. Emotional disorders and maladaptive eating patterns may also contribute to weight gain. Although patients frequently believe that their weight gain must be due to a "glandular problem," endocrine disorders are infrequently identified. While a thorough history (including dietary history) and physical are appropriate for all patients presenting with obesity, endocrine studies should be reserved for patients with features suggestive of a primary endocrine disorder (see Table 137.2).

Protein–calorie malnutrition is present in an alarming number of seriously ill hospitalized patients, with a prevalence as high as 50% in some surveys. Malnutrition may contribute to poor wound healing, decreased immune competence with increased susceptibility to sepsis, hypoventilation, decreased tolerance of chemotherapy, and delayed ambulation.

Many illnesses predispose patients to malnutrition. The most common mechanism is decreased nutritional intake that may be related to appetite loss associated with illness and drugs, surgical procedures, gastrointestinal disorders, swallowing problems, and impaired ability to self-feed secondary to neurologic and psychiatric illness. In addition, illnesses such as malabsorption, protein-losing enteropathies, diabetes, and nephrotic syndrome may result in increased loss of nutrients. The hypermetabolic state associated with major illness can also increase protein–calorie requirements. Cancers may cause malnutrition by any of these mechanisms and, in addition, may have independent effects on metabolic processes.

Marasmus and kwashiorkor-like syndromes may be present in malnourished patients. Marasmus is seen with prolonged starvation, when total dietary intake is inadequate. It is often seen with severe chronic illnesses, and these patients are generally recognizable by their weight loss, muscle wasting, loss of fat deposits, and general cachectic appearance. Kwashiorkor-like malnutrition occurs when the diet contains ample calories but little protein (e.g., prolonged administration of dextrose-containing intravenous fluids). In acute depletion, obesity may be present, and skeletal mass may be only slightly depleted. Laboratory tests of visceral proteins and immune competence are usually depressed.

Table 137.2
Organic Causes of Obesity

Drugs	Phenothiazines, tricyclic antidepressants, corticosteroids and oral contraceptives may be associated with weight gain.
Hypothyroidism	Rare cause of obesity; routine screening not indicated; weight gain is primarily fluid; other features present.
Cushing's syndrome	Truncal obesity, purple striae, "moon" facies, "buffalo hump," hypertension, glucose intolerance.
Stein-Leventhal syndrome	Oligomenorrhea, hirsutism; may be due to ovarian steroid secretion.
Insulin	Insulinoma or secondary to excessive exogenous insulin administration.
Hypogonadism	Particularly with postpubertal castration.
Hypothalamic	Secondary to inflammation, trauma, or tumors (particularly craniopharyngioma) affecting ventromedial nucleus.
Other neurologic causes	Pseudotumor cerebri, empty sella syndrome, hyperostosis frontalis interna.
Congenital syndromes	Adiposogenital dystrophy; Prader-Willi, Laurence-Moon-Biedl, Alstrom's, Albright's syndrome.
Abnormalities of fat distribution	Partial lipodystrophy, multiple lipomatosis, adiposis dolorosum.

Malnutrition may also be seen in outpatients who are chronically ill or alcoholic, as well as in some elderly individuals and children on inadequate diets. Anorexia nervosa and other eating disorders may result in life-threatening malnutrition.

Stature

The decision to investigate a child with short stature is determined more by the pattern of growth than by the current centile. A low growth velocity must always be investigated as a previously normal child may stop growing for several years before falling below the 3rd percentile. Poor growth is a nonspecific sign of disease.

Most children with short stature do not suffer from endocrinological or genetic disorders, but represent the bottom 3% of the normal distribution of height. These are instances of *hereditary short stature*. Many of these children have parents with short stature, and there are growth charts that take parental height into account.

Nutritional deficiency and hypoxia are common causes of short stature and may be associated with chronic pulmonary, cardiac, gastrointestinal, renal, or metabolic disease, as well as with a poor environment.

Chromosomal abnormalities are usually associated with characteristic features. Down's syndrome (trisomy-21) is characterized by growth retardation, mental deficiency, cardiac abnormalities, and a flat face with a short nose and epicanthic skin folds. Turner's syndrome (XO karyotype) is associated with a female phenotype, short stature, sexual infantilism, and characteristic facies. Turner variants may have short stature without the usual stigmata.

The *skeletal dysplasias* are characterized by short stature with abnormal skeletal proportions. They include a large variety of disorders; a complete discussion is beyond the scope of this chapter. Achondroplasia is the most common dysplasia (1 in 25,000 births). It is an autosomal dominant trait, but 80% of cases represent new mutations. These individuals have proximal limb shortening, short stature, and large heads with frontal bossing. Hurler's syndrome is a disorder of mucopolysaccharide distribution with associated skeletal abnormalities, a large skull, and typical facies.

Dysmorphic dwarfism includes a variety of syndromes of unknown etiology with intrauterine and postnatal growth retardation. *Hypopituitary dwarfism* may result from primary pituitary disease, hypothalamic dysfunction (including cases secondary to psychosocial deprivation) or end-organ resistance to growth hormone. These children tend to have normal skeletal proportions for their age.

Hypothyroidism is characterized by complete growth cessation and infantile skeletal proportions. The other physical findings of hypothyroidism may be subtle. The most serious consequences result when thyroid deficiency occurs early in life.

Cushing's syndrome may be due to endogenous or exogenous steroids. The other clinical signs may be less prominent in children than in adults.

There are few pathologic causes of tall stature. Tall stature is less often a concern to parents and children than short stature; however, girls with *familial tall stature* and a predicted height of greater than six feet may be considered for treatment with estrogens.

Klinefelter's syndrome is associated with normal growth velocity, long legs, small testes, and poor mental performance. *Marfan's syndrome* is characterized by above-average height, long limbs, arm span greater than height, lower segment greater than the upper segment, ocular abnormalities, and aortic aneurysm. *Giantism* is caused by excess growth hormone secretion before epiphyseal closure. These individuals have rapid linear growth and later develop the coarse features of acromegaly.

Miscellaneous causes of tall stature include homocystinuria, total lipodystrophy, XYY karyotype, thyrotoxicosis, exogenous obesity, sexual precocity and virilization syndromes, cerebral giantism, and Beckwith–Wiedemann syndrome.

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